UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE
United States Patent and Trademark Office
Address: COMMISSIONER FOR PATENTS
P.O. Box 1450
Alexandria, Virginia 22313-1450
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/063,587	05/03/2002	Audrey Goddard	P3230R1C001-168	4546

30313 7590 06/27/2006

KNOBBE, MARTENS, OLSON & BEAR, LLP
2040 MAIN STREET
IRVINE, CA 92614

EXAMINER
BASI, NIRMAL SINGH

ART UNIT	PAPER NUMBER
1646	

DATE MAILED: 06/27/2006

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	10/063,587	GODDARD ET AL.	
	Examiner	Art Unit	
	Nirmal S. Basi	1646	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *10 April 2006*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-5* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☐ Claim(s) *1-5* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date *4/10/06*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

Continued Examination Under 37 CFR 1.114

1. A request for continued examination under 37 CFR 1.114 was filed in this application after appeal to the Board of Patent Appeals and Interferences, but prior to a decision on the appeal. Since this application is eligible for continued examination under 37 CFR 1.114 and the fee set forth in 37 CFR 1.17(e) has been timely paid, the appeal has been withdrawn pursuant to 37 CFR 1.114 and prosecution in this application has been reopened pursuant to 37 CFR 1.114. Applicant's submission filed on 4/10/06 has been entered.

Response to Arguments

2. The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Claim Rejections - 35 USC § 101/ 112, First Paragraph

3. Claims 1-5 remain rejected under 35 U.S.C. 101 because the claimed invention is not supported by either a specific and substantial asserted utility or a well established utility for the reasons set forth below and in the previous office action and Examiners Answer.

Claims 1-5 remain rejected under 35 U.S.C. 112, first paragraph. Specifically, since the claimed invention is not supported by either a specific and substantial asserted utility or a well established utility for the reasons set forth below, in the previous Office action and Examiners Answer, one skilled in the art clearly would not know how to use the claimed invention.

Applicants' arguments under 35 USC 101 also support the request to withdraw the related rejection under 35 USC 112, first paragraph, enablement (p. 23-26 of response). As a result, both utility and enablement arguments will be discussed here.

Applicants argue the PTO has not met its burden of providing evidence showing that one of ordinary skill in the art would reasonably doubt the asserted utility. Applicants argue that the mRNA for the PRO1357 polypeptide is differentially expressed in stomach and lung tumors compared to normal stomach and lung tissue, respectively. The utility of the claimed invention is based on PRO1357 polypeptide being differentially expressed in stomach and lung tumors as compared to normal stomach and lung tissue, respectively. The making of the claimed antibodies to the differentially expressed polypeptides is argued to be useful as diagnostic tools, alone or in combination with other diagnostic tools.

Applicants argue that the patentable utility of the PRO1357 polypeptides is based on the PCR amplification data obtained from Example 18 in the specification. Example 18 measures the cDNA from different libraries using specific oligonucleotide probes. The polynucleotide data from Example 18 is argued to demonstrate that the polypeptide PRO135 and the antibodies that bind said polypeptide have utility as diagnostic markers for the presence of one or more cancerous tumors, but also serve as therapeutic targets for the treatment of these tumors. Applicants further argue the increase in PRO1357 mRNA levels also correlate with increased PRO1357 protein expression. It is noted for the record that the specification provides no protein expression data. There is no indication that protein expression was determined.

Therefore, the fundamental question is, does the data obtained from Example 18 in the specification measuring cDNA levels provide utility for the claimed PRO1357 antibodies? Applicants' arguments have been fully considered but are not found persuasive. In their response Applicants have provided 156 references some directly related to establishing a link between mRNA levels to protein expression, and some not. The office has supplied a sampling of references that show that there is unpredictability in establishing a link between an increase in mRNA levels and the corresponding increase in the encoded polypeptide. In response to Applicants arguments the office supplies, below, more references that support Examiners arguments. If PRO1357 polypeptide levels had been determined then there would be no disagreement, we would know the answer. Since the specification does not disclose that PRO1357 polypeptide levels were determined the art must be used to provide the answer. The problem is that the art provides arguments both ways, some references show a correlation between certain proteins others do not. There is no disclosure in the art or specification as to predict which polypeptides should show a correlation between increases mRNA and its encoded polypeptide. The only conclusion that can be reached based on the conflicting literature is that we have confusion and unpredictability and we cannot say that an increase in cellular PRO1357 mRNA levels results in an increase in cellular levels of PRO1357 polypeptide. The Office could also provide, if not 159 references as supplied by Applicants, a very high number arguing away from a correlation between mRNA and protein, but that would be futile. Does the one that supplies the most references wins? The point is the art provides data supporting both

arguments. There is no evidentiary support in the Declaration of Dr. Polakis that it remains a central dogma in molecular biology that increased mRNA levels are predictive of corresponding increased levels of the encoded polypeptide.

Further, based on the following references, the Examiner disagrees with the statement that enhanced mRNA levels are, as a general rule, indicative of enhanced levels of the encoded protein. For example, the textbook Genes VI (1997) by Benjamin Lewin points out that control of gene expression can occur at multiple stages and that production of RNA cannot inevitably be equated with the production of protein. More recently, Greenbaum et al. (Genome Biology, 2003, Vol. 4, Issue 9, pages 117. 1-117.8) cautions against assuming that mRNA levels are generally correlative of protein levels. The reference teaches (page 117.3 2nd column) that primarily because of a limited ability to measure protein abundances, researchers have tried to find correlations between mRNA and the limited protein expression data, in the hope that they could determine protein abundance levels from the more copious and technically easier mRNA experiments. To date, however, there have been only a handful of efforts to find correlations between mRNA and protein expression levels, most notably in human cancers and yeast cells. And, for the most part, they have reported only minimal and/or limited correlations. The reference further teaches (page 117.4, 2nd column) there are presumably at least three reasons for the poor correlations generally reported in the literature between the level of mRNA and the level of protein, and these may not be mutually exclusive. First, there are many complicated and varied post-transcriptional mechanisms involved in turning mRNA into protein that are not yet sufficiently well

defined to be able to compute protein concentrations from mRNA; second, proteins may differ substantially in their *in vivo* half lives; and/or third, there is a significant amount of error and noise in both protein and mRNA experiments that limit our ability to get a clear picture. The reference further notes (page 117.6, 2nd column) that to be fully able to understand the relationship between mRNA and protein abundances, the dynamic processes involved in protein synthesis and degradation have to be better understood. Hence, due to the teachings above, it would appear that production of mRNA, as a general rule, would not inevitably be predictive of equivalent levels of protein.

Applicants argue that Hu et al does not show a lack of correlation between mRNA and protein expression. This has been fully considered but is not found to be persuasive. The asserted utility for the polypeptide is based on the presumption that increased mRNA production leads to increased protein production. Hu et al. is directly on point by showing that the presumption is incorrect when designating proteins as diagnostic markers for cancer. Hu et al. (2003, Journal of Proteome Research 2:405-412) analyzed 2286 genes that showed a greater than 1-fold difference in mean expression level between breast cancer samples and normal samples in a microarray (p. 408, middle of right column) and discovered that, for genes displaying a 5-fold change or less in tumors compared to normal, there was no evidence of a correlation between altered gene expression and a known role in the disease. However, among genes with an l0-fold or more change in expression level, there was a strong and significant correlation between expression level and a published role in the disease (see discussion section). The instant specification does not disclose that PRO1357 mRNA

levels are expressed at l0-fold or higher levels compared with normal, matched tissue samples. Therefore, the skilled artisan would not reasonably expect that PRO1357 polypeptide could be used as a cancer diagnostic. Since the asserted utility for the claimed polypeptides is not in currently available form, the asserted utility is not substantial.

Applicants state the disclosed data support the over expression of PRO1357 polynucleotide and that a further correlation exists between the mRNA level and polypeptide levels. Contrary to Applicants' arguments no correlation between the PRO1357 mRNA levels and PRO1357 polypeptide level has been disclosed in the specification. PRO1357 polypeptide has not been shown to be over expressed in tumor tissue nor has the functionality of the PRO1357 polypeptide disclosed.

A review of the literature indicates that some references demonstrate a positive correlation between mRNA expression and protein levels, while some show no correlation. From this, one of ordinary skill in the art would not assume that if an mRNA were differentially expressed, the protein would also be expressed in a corresponding manner. A paper showing poor correlation is Anderson et al., Electrophoresis, Vol. 18, pages 533-537, 1997, who found that there was a poor correlation (0.48) between mRNA and protein levels in liver cells (abstract, page 535). They suggest that the two major phases of gene expression regulation (transcription through message degradation on the one hand, and translation through protein degradation on the other) are of approximately equal importance in determining the net output of proteins (page 536, left column). Anderson et al. also reanalyzed the set of data for plasma proteins

secreted by the liver that was published by Kawamoto et al., (Gene, 1996, Vol. 16, pages 1977-1981), in which the mRNA-to-protein relationship for nine plasma proteins was 0.96. However, when albumin (which is well-separated from the cluster of the remaining eight and thus exercises a disproportionate influence on the correlation coefficient) was omitted from the calculation, the correlation coefficient is reduced to – 0.19, which suggests a very poor correlation (page 536, right column). Lian et al., (2001, Blood 98:513-524) show a lack of correlation between mRNA expression and protein expression in mouse cells (see p. 514, top of left column: "The results suggest a poor correlation between mRNA expression and protein abundance, indicating that it may be difficult to extrapolate directly from individual mRNA changes to corresponding ones in protein levels."). See also Fessler et al., (2002, J. Biol. Chem. 277:31291-31302) who found a"[p]oor concordance between mRNA transcript and protein expression changes" in human cells (p. 31291, abstract). The evidence as a whole clearly indicates that one skilled in the art would not assume that an increase in mRNA levels results in increased protein levels without doing the empirical experimentation necessary to measure protein levels. The requirement for such empirical experimentation indicates that the asserted utility for the polypeptides is not substantial it is not in currently available form.

Further based on the Polakis declaration, it is disclosed that in approximately 20% of the observations an increases in the level of a particular mRNA does not correlate with changes in the level of protein expressed from that mRNA. Therefore,

further experimentation is required to confirm if the expression of PRO1357 nucleic acid results in increased levels of the encoded protein.

Based on the art it appears that the analysis in Example 18 is a very good starting point but more experimentation is required to establish utility under 35 USC 101. The art echoes the same sentiments that predictions of functionality based on Example 18 are not iron clad, in many cases overexpression of a specific nucleic acid cannot be associated with cancer. In addition, the references cited in prior Office Actions and those cited in this office Action suggest more experimentation is required to establish utility under 35 USC 101 for the claimed invention. Applicants are holding the publications cited by Examiner to higher standards than their own when arguing their drawbacks. The references cited in this Office Action supplement Examiners arguments that predicting function based on the data in Example 18 has flaws and will undermine the accuracy of the conclusions.

In conclusion Examiner appreciates the opinions of the Applicants' and the Declarations submitted by Dr. Grimaldi and Dr. Polakis. Taken as a whole, based on the numerous teaching in the art which caution on basing association of a disease with micoarray results and those references that caution against assuming that mRNA levels are generally correlative of protein levels alone, the claimed invention requires further research to establish utility. Just like Dr. Grimaldi and Dr. Polakis the authors cited by Examiner to support unpredictability in the art are also experts in the field. Therefore, further experimentation is required to confirm if the expression of PRO1118 nucleic acid

results in increased levels of the encoded protein and a tumor state. Thus, applicant's arguments have not been found persuasive, and the rejection is maintained.

Further, the polypeptide itself was not evaluated in the specification for actual expression in tissues. Since the encoding mRNA is expressed in stomach and lung tissue, one would reasonably expect the encoded protein also to be expressed, though at what levels it would be expressed is unknown. The protein does not have a recognized/characterized physiological/biochemical property. Proteins not identical to SEQ ID NO:78 have not been shown to exist in nature, let alone in stomach or lung tissue. As to the state of the prior art, other encoding nucleic acids usable for tumor markers have been identified, though none as a tumor marker were identical or highly similar to SEQ ID NO:77. Therefore, the connection of SEQ ID NO:77 to tumors was not known. The prior art is silent with respect to activity of PRO1357 or its relationship to a family of proteins with conserved structure and function. While the skill in the art for differential screening of nucleic acids has existed for over a decade, interpretation of the results depends, for example, on relative or absolute levels of the difference(s), the ability to generalize to more than one cell culture or tumor type or, conversely, the ability to pinpoint a particular tumor type (*e.g.*, adenocarcinoma *versus* squamal), and repeatability of the differential expression both in terms of frequency/prevalence and quantity/sensitivity. Further, there is evidence in the prior art that even for those nucleic acids differentially expressed in tumors, a correlated expression for the encoded protein is not a given. The breadth of the claims is broad, encompassing structural variation. There is very little guidance or direction about using the claimed polypeptide except that

the encoded nucleic acid of SEQ ID NO:77 is underexpressed in stomach and lung tumors. As discussed in previous Office actions, the specific type of tumor is not disclosed, nor are levels of expression, relative amounts or how many different tumor cDNA libraries from each tumor tissue were screened, for example. For all these reasons and those previous stated, it would require undue experimentation to use the invention as claimed.

The issue in this application is the insufficiency of disclosure to support a specific and substantial or well established utility or to allow the skilled artisan to use the claimed invention without undue experimentation. Because as previously discussed there is critical information lacking which includes: whether differences in nucleic acid expression of PRO1357 were significant, under what conditions differences could be detected, and what levels (relative or absolute) were detected in tumor and normal control, the skilled artisan cannot use (whether *in vivo* or *in vitro*) the claimed invention." As was stated above, and in the previous office Action the literature cautions researchers from drawing conclusions based on small changes in transcript expression levels between normal and cancerous tissue.

Applicants argue that polypeptides such as PRO1357 which are differentially expressed in certain cancers are useful as diagnostic tools. The argument has been fully considered, but is not persuasive. Were PRO1357 differentially expressed and were this expression significant, repeatable and the information sufficiently complete to allow use of the polypeptide without undue experimentation, it would have utility as a diagnostic tool. It, however, has none of these necessities. There is no showing or

reasonable expectation that PRO1357 is differentially expressed in certain cancers, even though its encoding nucleic acid of SEQ ID NO:77 appears to be underexpressed in stomach and lung tumors, though specifically which kind and at what levels is unknown.

Applicants argue that the data in Example 18 as discussed in the Declaration of Grimaldi demonstrates at least a two-fold difference in expression between normal and tumor tissues and the usefulness of the encoding nucleic acid as a diagnostic tool for determining the presence or absence of a tumor. The argument has been fully considered, but is not persuasive. The conclusionary statement of Grimaldi of the necessary existence of an at least two-fold differentiation in nucleic acid expression does not support a utility for or enable the invention because it does not fill important gaps in the disclosure needed to use the invention without significant further experimentation, such as expression level range for normal and tumor tissues, specific types of stomach or lung tumors detectable, and probability of detection for any particular stomach or lung tumor type (*e.g.*, whether one would reasonably expect underexpression in 10/10 or 1/20 tumors tested), or if and how much the PRO1357 polypeptide is expressed in normal *versus* tumor stomach and lung tissue. Even though the detection in Example 18 of the specification was carried out using cDNA libraries from tumor and normal tissue sample and, according to the declaration, the libraries were made from pooled samples of tissues, this does not fill the above discussed gaps. It is noted that Grimaldi in paragraph 6 of the declaration describes the detection as "semi-quantitative" and the specification for Example 18 as "standard quantitative". The

declaration also says (¶5) that "Data from a pooled sample are more likely to be accurate that data from a single individual." This begs the question of whether the tissue from an individual could be assessed for whether or not it is cancerous. Clinical diagnostics are not usually geared toward a populous but toward an individual's particular condition. While a "relative difference in expression between normal tissue and suspected cancerous tissue" can be informative, without more specifics about necessary sample size, expression level range for normal and tumor tissues, types of stomach or lung tissue that can used, and other questions, the specification has not provided the invention in an enabling form. Therefore, even accepting Dr. Grimaldi's opinion (see first paragraph of p. 13 of response), the declaration is insufficient to overcome the rejections of the claims under 35 USC 101 or 112, first paragraph, for the reasons discussed above.

Applicants argue that the report of Haynes et al. and Gygi et al. (Mol. Cell. Biol., 1999) do not support the Examiner's position that mRNA levels do not correlate with protein levels, pointing out that Haynes did not look at *single* genes and corresponding protein level. The argument has been fully considered, but is not persuasive. A complete reading of Haynes and Gygi et al. continues to support the reliance on Haynes et al. Applicants' point to the correlation coefficient of 0.935 in Haynes et al., saying that this shows a correlation instead of the lack of one. However, a full reading of Haynes et al. clarifies the data (p. 1726, first full paragraph):

> For the entire group (106 genes) for which a complete data set was generated, there was a general trend of increased protein levels resulting from increased mRNA levels. The

> Pearson product moment correlation coefficient for the whole data se (106 genes) was 0.935. This number is highly biased by a small number of genes with very large protein and message levels. A more representative subset of the data is shown in the inset of Fig. 5. It shows genes for which the message level was below 10 copies/cell and includes 69% (73 of 106 genes) of the data used in the study. The Pearson product moment correlation coefficient for this data set was 0.356.

Contrary to Applicants' assertion that Figures 5 and 6 of Gygi support the correlation of mRNA and protein levels, Gygi et al. show in Figure 5 the same figure as Fig. 1 of Haynes and show in Fig. 6, what is described for the Pearson correlation coefficients in the cited paragraph above. Gygi et al. say beginning in the last sentence in col. 1 of p. 1727 that, "The observed level of correlation between mRNA and protein expression levels suggest the importance of posttranslational mechanisms controlling gene expression. Such mechanisms include translational control .. and control of protein half-life.... Since these mechanisms are also active in higher eukaryotic cells, we speculate that there is no predictive correlation between steady-state levels of mRNA and those of protein in mammalian cells." As to correlation of an individual gene, Gygi et al. and Haynes et al. point to a great unpredictability about expression of a nucleic acid and its encoded protein. Predicting a correlation for any single gene is more difficult than for a large pool of genes showing a general trend. This can be seen by the low 0.356 correlation coefficient described above by Haynes et al. Each point in the figures of Haynes et al. and Gygi et al. are individual genes (see Fig. 1 and Figs. 5-6, respectively). Therefore, the authors did examine single genes. Haynes et al. supports the rejections of record and also says that the results are expected to be representative for mammalian cells (*e.g.*, like the human cell from which the PRO1357 nucleic acid

was isolated).

Other references have been reviewed here and in previous Office actions, which support the unpredictability by examining not just one, but a hundred or hundreds of mRNAs and corresponding proteins.

Applicants argue that the declarations of Grimaldi and Polakis support the teachings in Molecular Biology of the Cell, Genes VI, and Zhingang et al. (2004), that it is generally accepted that mRNA and protein expression are positively correlated. The argument has been fully considered, but is not persuasive. As discussed above, there is sound supporting evidence showing the unpredictability of saying level of expression of a particular nucleic acid will correlate with expression of the encoded protein. The argument of correlation between nucleic acid and protein expression has been previously addressed. Zhingang find that a correlation between mRNA and protein expression for the PSCA nucleic acid examined occurred in 93% of the samples so that it may be a promising diagnostic marker. There is no requirement for utility that a 100% correlation be present. Nevertheless, in the instance application, we have no correlation. There is no suggestion of multiple tumors tested. There are just "cDNA libraries isolated from different human tumor and normal human tissue samples." The declaration of Grimaldi says these samples were pooled samples. No relative or absolute values of expression for protein or nucleic acid were given in the specification. As discussed above, it is not clear whether one would reasonably expect underexpression in 10/10 or 1/20 tumors tested for the PRO1357 nucleic acid and/or

protein. If Zhingang et al. had obtained only a 5% correlation, it is doubtful he would have concluded that the nucleic acid would be a promising molecular marker.

Applicants have acknowledged on page 21 of their arguments that the correlation between changes in mRNA level and protein level is not exact, and there are exceptions. Considering the evidence as a whole, the specification, references supplied by applicant and those cited by examiner during the prosecution of this application further experimentation is required to establish utility.

Claim Rejections - 35 USC § 102

4. Claims 1-5 remain rejected under 35 U.S.C. 102(b) as being anticipated by WO 01/16318 or WO 00/12708 for the reasons set forth in the previous Office action.

Applicants argue that the instant application receives an effective filing date of 08/24/00 because the data of Example 18 was disclosed therein. The argument has been fully considered, but is not persuasive. Because the claims do not meet the requirements of 35 U.S.C. § 112, first paragraph, as discussed above, and the earlier application likewise do not meet those requirements, the instant application does not receive benefit of priority to earlier filed applications. Even though SEQ ID NO:77 and 78 and the expression information of Table 18 were previously disclosed, enablement thereof has not been established as discussed above.

5. This application contains sequence disclosures that are encompassed by the definitions for nucleotide and/or amino acid sequences set forth in 37 CFR 1.821(a)(1) and (a)(2). However, this application fails to comply with the requirements of 37 CFR 1.821 through 1.825 for the reason(s) set forth below or on the attached Notice To Comply With Requirements For Patent Applications Containing Nucleotide Sequence And/Or Amino Acid Sequence Disclosures. The application does not contain a paper copy of the sequence listing. Compliance with the sequence rules is required.

Applicant must comply with the sequence rules, 37 CFR 1.821 - 1.825. Failure to comply with these requirements will result in ABANDONMENT of the application under 37 CFR 1.821(g).

7. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Nirmal S. Basi whose telephone number is 571-272-0868. The examiner can normally be reached on 9:00 AM-5:30 PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Gary Nickol can be reached on 571-272-0835. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

Nirmal S. Basi
Art Unit 1646
6/22/06

GARY B. NICKOL, PH.D.
PRIMARY EXAMINER